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and joints (somatosensory system). The fourth system is visceral detection of gravity, upright position, and acceleration (meaning change in speed or direction of movement) by *visceral graviceptors*. These include stretch receptors in mesenteries or other connective tissue supporting organs or great vessels, and integrated systems of pressure detection in vessels and organs.[45] Such receptors have been localized to the kidneys and to the great vessels or their supporting structures in the mediastinum.[46] Mittelstaedt shows (by clever calculation and experimentation with people positioned in various ways on spinning centrifuge tables in the dark) that the visceral graviceptors control about 60% of our perception of position relative to gravity (meaning our sense of whether we are vertical or horizontal, or somewhere in between), compared to a 40% contribution made by the otolith organs.[47] Von Gierke (an older dean of vibration studies for the US space program) considers an inter-modality sensory conflict related to phase differences between the abdominal visceral graviceptors and the otolith organs to be a possible cause of motion sickness.[48]

The second critical element is central processing: how sensory information about motion and position is integrated by the brain, what other brain centers are activated, and what kinds of signals the brain then sends back to the body. Balaban and colleagues describe how the parabrachial nucleus network receives motion and position information from visual, vestibular (inner ear), somatosensory, and visceral sensory input, and is linked to brain

[45] Balaban and Yates 2004.

[46] Vaitl D, Mittelstaedt H, Baisch F. 2002. Shifts in blood volume alter the perception of posture: further evidence for somatic graviception. Int J Psychophysiol 44(1): 1–11.

[47] Mittelstaedt 1999.

[48] von Gierke HE, Parker DE. 1994. Differences in otolith and abdominal viscera graviceptor dynamics: implications for motion sickness and perceived body position. Aviat Space Environ Med 65(8): 747–51.

centers and circuits that mediate anxiety and fear, including the amygdala (a key mediator of fear reactions) and serotonin and norepinephrine-bearing neurons radiating from the midbrain.[49–51] Meaning that our sense of balance and stability in space is closely connected—neurologically—to fear and anxiety.

Balaban illustrates with a story. He asks the reader to visualize waiting in traffic on a hill for a light to turn. Out of the corner of your eye you see the truck next to you starting to inch forward, and you jam your foot on the brake, since your sensory system has told you that you are starting to slip backwards. There's a bit of panic in that moment, quickly settled as you realize you are indeed stable in space and not moving. The story illustrates how a sensation of unexpected movement elicits alerting and fear. When the sense of movement is ongoing and cannot be integrated with the evidence of the other senses, as happens in vertigo, there is a more prolonged fear reaction. In fact, as Balaban shows, the association of fear with vertigo has been known since ancient times.[52]

The third critical element is integrated neurologic outflow to the body from the parabrachial nucleus network to both the somatic (conscious, voluntary) and visceral (autonomic) effector systems. The somatic musculature is responsible for that fast foot on the brake, for righting movements of limbs, torso, and neck, and for breathing motions of the diaphragm and chest wall. The autonomic system is responsible for blood flow, heart rate, blood pressure, sweating, nausea, and other automatic, non-conscious modifications to visceral functioning. In a fear response, there is integrated outflow to these two systems—the somatic and visceral/

[49] Balaban and Thayer 2001.

[50] Balaban 2002.

[51] Halberstadt and Balaban 2003.

[52] Balaban and Thayer 2001.

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autonomic. Experimental work with animals shows that vestibular signaling has profound effects on autonomic regulation of body temperature, heart rate, vascular resistance, and circadian rhythms of activity and hormone secretion.[53,54] These effects extend to humans. Vestibular stimulation by passive linear acceleration causes blood pressure and heart rate increases, with diminished responses in people with reduced vestibular function.[55]

The parabrachial nucleus network is also involved in aversive learning,[56] an experience in which nausea, if present, plays a dominant role.[57]

In VVVD, subjects detect unusual types of movement (pulsation, internal vibration, internal quivering) or other sensations (pressure, a sense of fighting something to breathe, pins and needles) in the chest or in the coordinated chest-abdominal internal space. The chest and abdomen are separated and unified by the diaphragm, which, as a striated somatic muscle, has fine-grained sensitivity to motion and stretch. The diaphragm sends signals to the brain which are specific and localizable in time and space, as opposed to visceral receptors, which send signals that are vague, like discomfort, malaise, fullness, or nausea. The diaphragm is tightly

[53] Murakami DM, Erkman L, Hermanson O, Rosenfeld MG, Fuller CA. 2002. Evidence for vestibular regulation of autonomic functions in a mouse genetic model. Proc Natl Acad Sci USA 99(26): 17078–82.

[54] Wilson TD, Cotter LA, Draper JA, Misra SP, Rice CD, Cass SP, Yates BJ. 2006. Vestibular inputs elicit patterned changes in limb blood flow in conscious cats. J Physiol 575(2): 671–84.

[55] Yates BJ, Aoki M, Burchill P, Bronstein AM. 1999. Cardiovascular responses elicited by linear acceleration in humans. Exp Brain Res 125: 476–84.

[56] Balaban and Thayer 2001.

[57] Garcia J, Ervin FR. 1968. Gustatory-visceral and telereceptor-cutaneous conditioning: adaptation in internal and external milieus. Commun Behav Biol 1: 389–415.

bound to one of the largest abdominal organs, the liver, and they move as a unit during breathing.

The chest, via the mouth, nose, trachea, smaller airways, and air sacs of the lungs, is open to the air. Pressure fluctuations in the air (sound waves) have free access to this airspace within the body when we breathe. Pressure fluctuations in the air also have access to the ear, which is designed to funnel them to the tympanic membrane, which concentrates their energy and transmits it to the inner ear. The ear and the chest are different size spaces with walls of different mobility and elasticity. Hence they respond differently to air pressure fluctuations (sound waves) of different sizes.

Studies of whole-body vibration focus on the easily mobile diaphragm and coupled abdominal organs. Being mobile, with the air of the lungs on one side and the soft abdominal wall on the other, this thoraco-abdominal system is easily set in motion by lower energy (amplitude) vibrations than are required to perturb other parts of the body.[58] Each part of the body has its own resonance frequency with regard to vibration. When an object is vibrated at its resonance frequency, the vibration is amplified. The resonant frequency of the thoraco-abdominal system, as it moves vertically towards and away from the lungs, lies between 4 and 8 Hz for adult humans.[59] Vibrations between 4 and 6 Hz set up resonances in the trunk with amplification up to 200%.[60] Related chest and abdominal effects are found in the same frequency range. Vibrations in the 4–8 Hz range influence breathing movements, 5–7 Hz can cause chest pains, 4–10 Hz abdominal pains, and 4–9

[58] Coermann RR, Ziegenruecker GH, Wittwer AL, von Gierke HE. 1960. The passive dynamic mechanical properties of the human thorax-abdominal system and of the whole body system. Aerosp Med 31(6): 443–55.

[59] von Gierke and Parker 1994.

[60] Hedge 2007.

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Hz a general feeling of discomfort.[61] In small children under 40 pounds, the vertical resonance or power absorption peaks at 7.5 Hz, as opposed to 4–5 Hz for adults.[62]

Low frequency noise can cause the human body to vibrate, as quantified by researchers in Japan.[63] The degree to which the body surface is induced to vibrate by low frequency noise is correlated with subjective unpleasantness (a sensation suggesting visceral as well as surface/somatic stimulation by the noise).[64]

With this background, I propose the following mechanism for VVVD. Air pressure fluctuations in the range of 4–8 Hz, which may be harmonics of the turbine blade-passing frequency, may resonate (amplify) in the chest and be felt as vibrations or quivering of the diaphragm with its attached abdominal organ mass (liver). Slower air pressure fluctuations, which could be the blade-passing frequencies themselves or a low harmonic (1–2 Hz), would be felt as pulsations, as opposed to the faster vibrations or quivering. (The vibrations or pressure fluctuations may also be occurring at different frequencies, without this particular resonance amplification.) The pressure fluctuations in the chest could disturb visceral receptors, such as large vessel or pulmonary baroreceptors or mediastinal stretch receptors which function as visceral graviceptors. These aberrant signals from the visceral graviceptors, not concordant with signals from the other parts of the motion-detecting system, have the potential to activate

[61] Rasmussen 1982.

[62] Giacomin J. 2005. Absorbed power of small children. Clin Biomech 20(4): 372–80.

[63] Takahashi Y, Yonekawa Y, Kanada K, Maeda S. 1999. A pilot study on the human body vibration induced by low-frequency noise. Ind Health 37: 28–35.

[64] Takahashi Y, Kanada K, Yonekawa Y, Harada N. 2005. A study on the relationship between subjective unpleasantness and body surface vibrations induced by high-level low-frequency pure tones. Ind Health 43: 580–87, p. 580.

the integrated neural networks that link motion detection with somatic and autonomic outflow, emotional fear responses, and aversive learning. The people who are susceptible to responding in this way are those who in the past have become nauseated in response to other vertically oriented, anomalous environmental movements (seasickness or carsickness). Thus panic episodes with autonomic symptoms such as tachycardia and nausea arise during wakefulness or sleep in people with pre-existing motion sensitivity but without prior history of panic, anxiety, or other mental health disorders. Repeated triggering of these symptoms creates aversive learning, wherein the person begins to feel horror and dread of things associated with the physical sensations, such as his bedroom or house where he previously found comfort and regeneration.

VVVD was identified in the study in 14 out of 21 adult subjects. The behavior and experiences of other subjects, especially children, could be interpreted as partial manifestations of the same problem. For example, the two toddlers in the study, both age 2½ (A3, G5), had night terrors. They awoke screaming multiple times per night, and were inconsolable and difficult to get back to sleep. The little girl (G5) would fight her mother, grabbing onto the posts of the bunk bed, to avoid going back into her own bed after awakening in this state. This shows clear parallels with the fear responses, prolonged awake periods, and aversive responses of the adults with VVVD. Both toddlers were agitated and irritable in the daytime, also similar to the adults in the study. Both 5-year-olds in the study, a boy and a girl (C7, G4), also frequently woke up fearful at night.

Perturbing the inner ear

I propose that disrupted stimulation of other channels of the balance system, especially the inner-ear vestibular organs, is also likely to play a role in Wind Turbine Syndrome. Altogether, in subjects with or without VVVD, the Wind Turbine Syndrome core symptoms resemble the symptoms of a balance or vestibular

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disorder, meaning malfunctioning of the inner-ear motion-detecting organs (peripheral vestibular dysfunction) or of brain processing of balance-related neural signals (central balance dysfunction). These symptoms may arise near wind turbines due to abnormal stimulation of the classical balance pathways (visual, vestibular, and somatosensory), perhaps in an additive fashion if several pathways are disturbed simultaneously.

A clinical rule of thumb is that two of the three classical balance channels have to be working and producing coherent information (with agreement among channels) for a person to keep his or her balance. (How this clinical rule will incorporate the new fourth channel of balance information is yet to be seen. It may be that the sensory integrative process is actually broader, taking into account the amounts and quality of information coming from each channel, not just whether a channel is active.) The three classical pathways are 1) vision, which includes a) seeing one's orientation relative to objects and the orientation of objects relative to gravity, b) movement of images across the retina, called "retinal slip," and c) parallax or distance detection; 2) somatosensory, which involves stretch signals from muscles, tendons, and joints, and touch sensations from the skin; and 3) signals from the inner-ear vestibular organs.

The vestibular organs are 1) the semicircular canals, which detect angular acceleration during rotation of the head in any of three planes, and 2) the otolith organs (utricle and saccule), which detect gravity, tilt (static or moving), and linear accelerations by virtue of microscopic calcium carbonate crystals (otoconia) positioned in a protein matrix over the sensing hair cells. In the utricle, the patch of hair cells plus otoconia (called the macula) is oriented horizontally and is sensitive to tilts and (in upright people) to the horizontal component of linear accelerations. In the saccule, the macula is vertical, sensitive to tilts and to the vertical component of linear

accelerations (including gravity) in upright people. The inner-ear or labyrinthine organs are delicate, membranous, interconnected structures with fluid inside (endolymph) and outside (perilymph), suspended in tiny canals and chambers through solid temporal bone at the base of the skull. The vertically oriented macula of the saccule is firmly bound to temporal bone over its entire area, but the horizontally oriented macula of the utricle has been recently found to be attached to temporal bone only at its anterior end,[65] a property that gives it an additional degree of freedom that may influence its tuning or resonance with regard to vibration.[66] Hair cells, which send neural signals when mechanically perturbed, are also present in specific parts of the semicircular canals and the cochlea, which is the spiral-shaped hearing organ.

In the current study, two subjects (C2, E2) were sensitive to the visual pathway with regard to triggering of symptoms. They developed severe headaches when exposed to the moving shadows of turbine blades. One (C2) had known migraine and was prone to vertigo. The other (E2) had fibromyalgia and a history of two pre-exposure episodes of vertigo. Fibromyalgia, a syndrome of chronic, diffuse pain of central origin,[67] is frequently accompanied by vertigo and dizziness.[68]

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[65] Uzun-Coruhlu H, Curthoys IS, Jones AS. 2007. Attachment of the utricular and saccular maculae to the temporal bone. Hear Res 233(1–2): 77–85.

[66] Todd NP, Rosengren SM, Colebatch JG. 2009. A utricular origin of frequency tuning to low-frequency vibration in the human vestibular system? Neurosci Lett 451(3): 175–80.

[67] Staud R, Cannon RC, Mauderli AP, Robinson ME, Price DD, Vierck CJ Jr. 2003. Temporal summation of pain from mechanical stimulation of muscle tissue in normal controls and subjects with fibromyalgia syndrome. Pain 102: 87–95.

[68] Rosenhall U, Johansson G, Orndahl G. 1996. Otoneurologic and audiologic findings in fibromyalgia. Scand J Rehabil Med 28(4): 225–32. In this study, 72% of 168 fibromyalgia patients had dizziness or vertigo, most with abnormalities on otoneurologic testing.

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Two subjects (C2, J2) noticed vibrations in their lower legs at certain locations on their properties, which opens the possibility of disruption of the somatosensory channel.[69] An audiologist detected vibration in the floor of an affected room in the C family's house, becoming nauseated when he put his forehead against it, an effect he interpreted as stimulation of the vestibular organs by bone conduction.[70]

I suspect that the inner-ear vestibular organs—and the cochlea—are abnormally stimulated in Wind Turbine Syndrome, especially in subjects who have marked ear symptoms such as tinnitus (including the sensation of noise inside the head) and ear pressure, popping, or pain. Families A and B, exposed to the same set of turbines, showed this pattern of symptoms especially strongly. All four adults (A1, A2, B1, B2) also had unsteadiness on their feet without accompanying vertigo or history of migraine, vertigo, prior unsteadiness, or neurologic disease. Unsteady gait, or ataxia, is generally associated with cerebellar dysfunction, but can also indicate otolith dysfunction.[71] (Vestibular nuclei in the brainstem are richly interconnected with the cerebellum.)[72] Other subjects (C2, G1, J1) had vertigo during exposure (C2 also had observed nystagmus), suggesting that disordered signals were reaching the vestibulo-ocular reflex arc from the semicircular canals or otolith organs.

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[69] Hanes DA, McCollum G. 2006. Cognitive-vestibular interactions: a review of patient difficulties and possible mechanisms. J Vestib Res 16(3): 75–91. Vibration of calf muscles is a method sometimes used in balance studies to simulate somatosensory disturbance, p. 77.

[70] Noise report prepared for family C, May 2006.

[71] Schlindwein P, Mueller M, Bauermann T, Brandt T, Stoeter P, Dieterich M. 2008. Cortical representation of saccular vestibular stimulation: VEMPs in fMRI. Neuroimage 39: 19–31.

[72] Colebatch JG, Halmagyi GM, Skuse NF. 1994. Myogenic potentials generated by a click-evoked vestibulocollic reflex. J Neurol Neurosurg Psychiatry 57(2): 190–97.

In Wind Turbine Syndrome, I hypothesize that low frequency noise or vibration impinges on the delicately mobile labyrinthine organs, but not in a way that stimulates the cochlea to a coherent representation of sound. Instead, the low frequency noise or vibration, I suggest, may stimulate various parts of the labyrinth in a disorganized fashion, experienced as tinnitus from the cochlea, a distorted sense of vertical from the otolith organs, or illusory self-motion from the otolith organs or semicircular canals. The dominant sensory impression may depend on 1) the frequencies and intensities of low frequency noise and vibration coming from the turbines, 2) whether the noise or vibration arrives at the ear through the air and outer/middle ear or is bone-conducted, and 3) the susceptibilities and prior histories of the subjects, such as migraine with its tendency towards vertigo, prior damage to the cochlea, or other conditions or anomalies of the inner ear.[73]

The statistical correlation in the current study between tinnitus and ear popping, pressure, or pain during exposure suggests a refinement to this mechanism: altered fluid pressure relationships in the inner ear may distort cochlear mechanics during exposure and cause tinnitus, and distort utricular and saccular mechanics to create instability or ataxia and other second-order vestibular symptoms.

Low frequency noise, in fact, is known to distort endolymphatic pressure and volume after just short exposures to loud but not

[73] For example, dehiscence of the superior semicircular canal, in which alterations in inner-ear pressure relationships due to a "third window" effect (from an abnormal hole in the bone between the superior semicircular canal and the cranial cavity) cause conductive hearing loss, increased sensitivity to bone-conducted sound or vibration, and the tendency to become unbalanced by sounds (Tullio effect). Dislocation of the stapes footplate, labyrinthine fistulas, and endolymphatic hydrops can also underlie the Tullio phenomenon. (See Colebatch JG, Day BL, Bronstein AM, Davies RA, Gresty MA, Luxon LM, Rothwell JC. 1998. Vestibular hypersensitivity to clicks is characteristic of the Tullio phenomenon. J Neurol Neurosurg Psychiatry 65: 670–78.)

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damaging low frequency tones.[74] This temporary effect is associated with hyperacusis, a distortion of hearing function in which sounds are perceived as louder.[75] One subject in the current study, G2, had hyperacusis while living near turbines, and another (C2) noticed hyperacusis after her tinnitus resolved, after she moved away from the turbines. Tinnitus may also be associated with increased perilymphatic and intracranial pressure in the presence of an open cochlear aqueduct, which provides a direct channel linking these two fluid spaces.[76]

There is both animal and human precedent for thinking that certain types of environmental noise or vibration may stimulate the otolith organs and cause disturbance to motion and position sense. Vestibular organ structures have been conserved during evolution, meaning they are rather similar in fish, amphibians, and other vertebrate taxa, including humans. All the vertebrates have semicircular canals and otolith organs. Like us, fish use their otolith organs (utricle, saccule, and an extra one, the lagena) to sense linear accelerations and tilt relative to gravity, but these organs in "non-specialist" fish species (such as cod) are also the fishes' auditory organs. The otolith organs in these fish are highly sensitive to nearby perturbations in the water ("near-field sound")[77] with peak sensitivities in the low frequency range between 40 and 120 Hz.[78] Atlantic cod otolith organs are so sensitive to

[74] Salt AN. 2004. Acute endolymphatic hydrops generated by exposure of the ear to nontraumatic low-frequency tones. J Assoc Res Otolaryngol 5(2): 203–14.

[75] Salt 2004.

[76] Reid A, Cottingham CA, Marchbanks RJ. 1993. The prevalence of perilymphatic hypertension in subjects with tinnitus: a pilot study. Scand Audiol 22: 61–63.

[77] Sand O, Karlsen HE, Knudsen FR. 2008. Comment on "Silent research vessels are not quiet" [J Acoust Soc Am 2007; 121(4): EL145–50]. J Acoust Soc Am 123(4): 1831–33.

[78] Fay RR, Simmons AM. 1999. The sense of hearing in fishes and amphibians. In *Comparative Hearing: Fish and Amphibians*, ed. Fay RR, Popper AN, pp. 269–317. Springer-Verlag, New York.

infrasound in water (at 0.1 Hz, or one wave every 10 seconds) that the fish may be able to use seismic sounds from the Mid-Atlantic Ridge or the sounds of waves breaking on distant shores, or even more complex mechanisms, to guide them during migration.[79,80] Directional infrasound detection plays a role in predator avoidance behaviors.[81]

In humans, there is a substantial body of experimental evidence showing that both air-conducted sound and bone-conducted sound (vibration) stimulate the otolith organs and cause measurable impacts on vestibular reflexes, independent of their stimulation of the cochlea. Air-borne sound in the form of loud clicks or short tone bursts induces inhibitory neural signals in the sternocleidomastoid muscles in the anterior neck. Called the *vestibular evoked myogenic potential* (VEMP), this is an extremely fast or "short-latency" neural response that is part of the vestibulo-collic reflex.[82] Bone-conducted sound or vibration is more efficient than air-conducted clicks or tones at stimulating the otolith organs: both the absolute decibel levels and decibels above hearing threshold needed to produce the VEMP response are lower for bone-conducted sound.[83]

Studies of both the VEMP and—a second measure of vestibular function—the *ocular vestibular evoked myogenic potential* (OVEMP) show that the tuning (best frequency response) for both

[79] Sand O, Karlsen HE. 1986. Detection of infrasound by the Atlantic cod. J Exp Biol 125: 197–204.

[80] Sand O, Karlsen HE. 2000. Detection of infrasound and linear acceleration in fishes. Phil Trans R Soc Lond B 355: 1295–98.

[81] Karlsen HE, Piddington RW, Enger PS, Sand O. 2004. Infrasound initiates directional fast-start escape responses in juvenile roach Rutilus rutilus. J Exp Biol 207(Pt 24): 4185–93.

[82] Colebatch et al. 1994.

[83] Welgampola MS, Rosengren SM, Halmagyi GM, Colebatch JG. 2003. Vestibular activation by bone conducted sound. J Neurol Neurosurg Psychiatry 74: 711–18.

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VEMP and OVEMP for air-conducted sound lies between 400 and 800 Hz.[84] Whereas with bone-conducted sound (vibration), the best frequency response for both VEMP and OVEMP is at 100 Hz. Modeling of the frequency tuning and other aspects of the response, such as laterality, phase differences, and gain, suggests that the air-conducted peak comes from the rigidly attached saccule, whereas the bone-conducted or vibratory peak derives from the more mobile utricle.[85] A particular type of vestibular hair cell, Type I cells, is thought to be involved in the utricular response and accounts for the marked sensitivity of the OVEMP response to vibration, since these cells typically produce a strong neural vestibular signal in response to a low degree of mechanical disturbance.[86,87]

Most exciting, Todd et al. provide direct experimental evidence that at the 100 Hz tuning peak, the vestibular organs (probably utricle, as above) of normal humans are *much more sensitive than the cochlea* to low frequency bone-conducted sound/vibration.[88] The researchers applied vibration directly to the skin over the bony mastoid prominence behind the subjects' ears, adjusting the power by measuring the tiny whole-head acceleration produced by each vibration force and frequency. They were able to elicit and measure neural signals of the vestibulo-ocular reflex (OVEMP, as above) at vibration intensities 15 dB below the subjects' hearing thresholds. In other words, the amount of vibration/bone-conducted sound was so small that the subjects could not hear it, yet the vestibular parts of their inner ears still responded to the vibration and

[84] Todd et al. 2009.

[85] Todd et al. 2009.

[86] Todd et al. 2009.

[87] Curthoys IS, Kim J, McPhedran SK, Camp AJ. 2006. Bone conducted vibration selectively activates irregular primary otolithic vestibular neurons in the guinea pig. Exp Brain Res 175(2): 256–67.

[88] Todd et al. 2008.

transmitted signals into the balance and motion networks in the brain, resulting in specific types of eye muscle activation. Since dB is a base 10 logarithmic measure, *15 dB below* means a signal 0.0316 ($10^{-1.5}$), or about 3%, of the power or amplitude of the signal these normal subjects could hear.

The researchers note that "the very low thresholds we found are remarkable as they suggest that humans possess a frog- or fish-like sensory mechanism which appears to exceed the cochlea for detection of substrate-borne low-frequency vibration and which until now has not been properly recognized."[89] Thus the potential exists, in normal humans, for stimulation of balance signals from the inner ear by low frequency noise and vibration, even when the noise or vibration does not seem especially loud, or even cannot be heard. In the presence of pre-existing inner-ear pathology, thresholds for vestibular stimulation by noise or vibration are even lower than in normal subjects.[90]

Central balance processing

When there is conflict in neurologically normal people among the signals coming from the different balance channels, the brain areas that integrate the information quickly compensate by suppressing or down-weighting information from the anomalous channel[91]—information that does not match what is coming from the other channels. On functional brain scans, vestibular and visual cortical areas show a pattern of inverse activation and deactivation, such

[89] Todd et al. 2008, p. 41.

[90] Colebatch et al. 1998. See footnote 73.

[91] Jacob RG, Redfern MS, Furman JM. 2009. Space and motion discomfort and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry 80(1): 74–78. E-pub 2008 July 24.

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that vestibular activation deactivates visual cortex and vice versa.[92,93] In people with vestibular organ damage, long-term compensation promotes reliance on vision ("visual dependence") or on somatosensory input from muscles, tendons, joints, and skin ("surface dependence"). A visually dependent vestibular patient cannot adequately suppress visual input and up-weight vestibular signals because of pre-existing problems with the vestibular channel,[94] leaving the person dependent on visual perception of motion and position even in environments where the visual information is ambiguous. When combined with the sense of fear generated by a feeling of postural instability or uncertainty (as reviewed above), this can create fear of heights.

It can also cause Space and Motion Discomfort,[95] a condition in which situations challenging to motion and position sense create discomfort. These situations include looking up at tall buildings, scanning shelves in a supermarket, closing eyes in the shower, leaning far back in a chair, driving through tunnels, riding in an elevator, riding in the back seat of a car, or reading in the car.[96]

Even without vestibular organ disease, some people have Space and Motion Discomfort due to a central or brain-based difficulty with

[92] Brandt T, Bartenstein P, Janek A, Dieterich M. 1998. Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. Brain 121(Pt. 9): 1749–58.

[93] Brandt T, Dieterich M. 1999. The vestibular cortex: its locations, functions, and disorders. Ann NY Acad Sci 871: 293–312.

[94] Redfern MS, Yardley L, Bronstein AM. 2001. Visual influences on balance. J Anxiety Disord 15(1–2): 81–94.

[95] Jacob RG, Woody SR, Clark DB, Lilienfeld SO, Hirsch BE, Kucera GD, Furman JM, Durrant JD. 1993. Discomfort with space and motion: a possible marker of vestibular dysfunction assessed by the Situational Characteristics Questionnaire. J Psychopathol Behav Assess 15(4): 299–324.

[96] Jacob et al. 2009. As a rural physician, I might also ask patients about driving past rows of parallel trees, especially with the low winter sun flashing between the trunks, as the rural equivalent of looking at lights on the wall of a tunnel.

the process of integrating balance signals into a coherent, moment-to-moment representation of their motion and orientation in space. Balance testing using posturography shows that such people have difficulty down-weighting anomalous information from either the visual or somatosensory channel, or have a mild, central disorder of balance control with increased postural sway even under non-challenging conditions.[97–99]

Space and Motion Discomfort is common in patients with anxiety disorders,[100,101] migrainous vertigo,[102] and migraine-anxiety related dizziness.[103] Vertigo is especially characteristic of migraine and may at times occur as a migraine aura with or without headache.[104] In one study, dizziness or vertigo was found in 54% of 200 migraine patients, half of whom also had a history of motion sickness, compared with 30% of people with tension-type headaches.[105] In a study of 72 patients with isolated recurrent vertigo, 61% were found to have migraine, compared to 10% in a control group of orthopedic patients.[106] Abnormal balance testing

[97] Redfern MS, Furman JM, Jacob RG. 2007. Visually induced postural sway in anxiety disorders. J Anxiety Disord 21(5): 704–16. NIH Public Access Author Manuscript, pp. 1–14.

[98] Jacob et al. 2009.

[99] Furman JM, Balaban CD, Jacob RG, Marcus DA. 2005. Migraine-anxiety related dizziness (MARD): a new disorder? J Neurol Neurosurg Psychiatry 76: 1–8.

[100] Jacob et al. 2009.

[101] Redfern et al. 2007.

[102] Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. 2001. The interactions of migraine, vertigo, and migrainous vertigo. Neurology 56: 436–41.

[103] Furman et al. 2005.

[104] Furman et al. 2005.

[105] Kayan A, Hood JD. 1984. Neuro-otological manifestations of migraine. Brain 107: 1123–42.

[106] Lee H, Sohn SI, Jung DK, Cho YW, Lim JG, Yi SD, Yi HA. 2002. Migraine and isolated recurrent vertigo of unknown cause. Neurol Res 24(7): 663–65.

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is seen in patients with migraine but not in those with tension-type headaches.[107] Balance testing shows that both central and vestibular organ balance problems are found in migraine patients, especially in those who experience dizziness or vertigo.[108]

About 50% of migraine sufferers in general have histories of motion sickness, compared to only about 20% in people with tension headaches.[109] Motion sickness is the most common vestibular symptom in migraine. Motion sickness is provoked by excessively moving environments (amusement park rides, boats in rough water, airplanes in turbulence, the back of a school bus) or situations of conflict among visual, vestibular, somatosensory, and visceral signals to the balance system (reading in the car, riding in the back seat, driving in snow, simulators, IMax movies, computer images and games, space travel). The nausea of motion sickness may be accompanied by dizziness, cold sweat, pallor, headache, increased salivation, sleepiness, and apathy or disinclination for physical or mental work, thus sharing many symptoms with migraine.[110] Like migraine, motion sickness is more common in women.[111] Visual migraine aura without headache is increased in adults with a history of childhood motion sickness. Motion sickness is not associated with peripheral vestibular disorders, however, such as benign paroxysmal positional vertigo, Meniere's disease, or vestibular neuritis.[112]

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[107] Ishizaki K, Mori N, Takeshima T, Fukuhara Y, Ijiri T, Kusumi M, Yasui K, Kowa H, Nakashima K. 2002. Static stabilometry in patients with migraine and tension-type headache during a headache-free period. Psychiatry Clin Neurosci 56(1): 85–90.

[108] Furman et al. 2005.

[109] Marcus DA, Furman JM, Balaban CD. 2005. Motion sickness in migraine sufferers. Expert Opin Pharmacother 6(15): 2691–97.

[110] Marcus et al. 2005.

[111] Marcus et al. 2005.

[112] Marcus et al. 2005.

The dizziness associated with anxiety disorders is not necessarily caused by the anxiety, as is often assumed in clinical practice, but may have a component of disturbed balance control.[113,114] For example, the presence of panic or fear of heights is significantly associated with abnormalities on caloric testing, a form of vestibular testing.[115] A positive result on a questionnaire for Space and Motion Discomfort is significantly associated with abnormality on posturography showing either surface[116] or visual[117] dependence. In testing of vestibulo-ocular reflexes, anxiety patients have been found to have higher vestibular sensitivity or gain than normal controls.[118] Balance assessments of patients diagnosed with panic attacks or agoraphobia (fear of leaving the house) show a high proportion with abnormalities of vestibular function, in some studies greater than 80%, especially if the patients have episodes of dizziness between panic attacks.[119–122]

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[113] Furman et al. 2005.

[114] Eckhardt-Henn A, Breuer P, Thomalske C, Hoffmann SO, Hopf HC. 2003. Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. J Anxiety Disord 17(4): 369–88.

[115] Jacob et al. 2009.

[116] Jacob et al. 2009.

[117] Redfern et al. 2007.

[118] Furman JM, Redfern MS, Jacob RG. 2006. Vestibulo-ocular function in anxiety disorders. J Vestib Res 16: 209–15.

[119] Perna G, Dario A, Caldirola D, Stefania B, Cesarani A, Bellodi L. 2001. Panic disorder: the role of the balance system. J Psychiatr Res 35(5): 279–86.

[120] Jacob RG, Furman JM, Durrant JD, Turner SM. 1996. Panic, agoraphobia, and vestibular dysfunction. Am J Psychiatry 153(4): 503–12.

[121] Yardley L, Britton J, Lear S, Bird J, Luxon LM. 1995. Relationship between balance system function and agoraphobic avoidance. Behav Res Ther 33(4): 435–39.

[122] Yardley L, Luxon LM, Lear S, Britton J, Bird J. 1994. Vestibular and posturographic test results in people with symptoms of panic and agoraphobia. J Audiol Med 3: 58–65.

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Thus problems with balance function can be due to abnormalities of the inner-ear vestibular organs (utricle, saccule, and semicircular canals) or to abnormal central (brain) integration of balance signals. Mild (mostly central) abnormalities are common and associated with common conditions such as migraine, motion sensitivity, vertigo, and several types of anxiety disorder. People with mild balance abnormalities only feel off balance or insecure in challenging situations where the available sensory information is inadequate or confusing, such as at heights or in the situations described in the questionnaire for Space and Motion Discomfort. The rest of the time, people with mild, compensated balance deficits feel normal and securely oriented in space.

However, if a person is already in a state of adaptation to an ongoing vestibular organ or central balance deficit—even mild, fully compensated deficits—he or she is at particular risk for decompensation with exposure to new balance challenges. Many of the affected people in the present study, I suspect, were in this condition, because their medical histories reveal a variety of risks for mild baseline balance dysfunction. These risks include motion sensitivity, migraine disorder, prior damage to inner-ear organs from industrial noise exposure or chemotherapy, autoimmune disease,[123] fibromyalgia,[124] and normal aging (over 50). We may also consider normal early childhood (age 1–4 or so) as a time of natural mild balance dysfunction[125,126] (see discussion at the end

[123] Rinne T, Bronstein AM, Rudge P, Gresty MA, Luxon LM. 1998. Bilateral loss of vestibular function: clinical findings in 53 patients. J Neurol 245(6–7): 314–21.

[124] Rosenhall U, Johansson G, Orndahl G. 1996. Otoneurologic and audiologic findings in fibromyalgia. Scand J Rehabil Med 28(4): 225–32.

[125] Foudriat BA, Di Fabio RP, Anderson JH. 1993. Sensory organization of balance responses in children 3–6 years of age: a normative study with diagnostic implications. Int J Pediatr Otorhinolaryngol 27(3): 255–71.

[126] Steindl R, Kunz K, Schrott-Fischer A, Scholtz AW. 2006. Effect of age and sex on maturation of sensory systems and balance control. Dev Med Child Neurol 48(6): 477–82.

of the next section). Other potential risks for chronic balance deficits, not seen in this study, are whiplash injury and head injury, including concussions and milder head impacts without loss of consciousness,[127–129] and chronic inner-ear conditions such Meniere's disease, dehiscence of the superior semicircular canal, and others.[130]

Cognition and vestibular function

It is now becoming apparent that a variety of cognitive functions depend on coherent vestibular signaling. Clinicians who work with balance-disordered patients are familiar with their struggles with short-term memory, concentration, multitasking, arithmetic, and reading.[131,132] In the perilymphatic fistula syndrome, for example (a form of inner-ear pathology that can follow whiplash, minor head injuries, or pressure trauma to the ear), symptoms of dizziness, headache, stiff neck, and disturbed sleep are accompanied by marked mental performance deficits compared to the patient's baseline.[133] Such cognitive symptoms are difficult to evaluate clinically and are often dismissed as psychological in origin.[134] However, recent research using imaging and other modalities shows that vestibular function exerts a powerful influence over human thinking and memory.

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[127] Grimm RJ, Hemenway WG, Lebray PR, Black FO. 1989. The perilymph fistula syndrome defined in mild head trauma. Acta Otolaryngol Suppl 464: 1–40.

[128] Ernst A, Basta D, Seidl RO, Todt I, Scherer H, Clarke A. 2005. Management of posttraumatic vertigo. Otolaryngol Head Neck Surg 132(4): 554–58.

[129] Claussen CF, Claussen E. 1995. Neurootological contributions to the diagnostic follow-up after whiplash injuries. Acta Otolaryngol Suppl 520, Pt. 1: 53–56.

[130] Colebatch et al. 1998.

[131] Hanes and McCollum 2006.

[132] Grimm et al. 1989.

[133] Grimm et al. 1989.

[134] Hanes and McCollum 2006.

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The vestibular system is ancient in the vertebrate lineage. Hence its neural connections ramify widely in both older and more recently evolved parts of the brain, including the brainstem, midbrain, cerebellum, and occipital, parietal, and frontal cortex.[135] Vestibular injury causes specific cognitive difficulties, but not general cognitive impairment.[136] Vestibular effects on cognition are often attributed to competing stimuli (meaning, challenges to movement and position sense draw attention away from cognitive tasks), but may actually reflect the direct dependence of certain cognitive operations on the vestibular system.[137]

Vestibular input is critical for spatial thinking, body and spatial awareness, spatial memory, and complex spatial or map calculations.[138] Dynamic, active vestibular signaling is needed during the acquisition, storage, and use of information with spatial components, such as building mental maps or deducing a novel path between two points.[139] Patients with 5–10 year histories of bilateral vestibular loss showed marked deficits in a classic experimental task of spatial memory and navigation, accompanied, on average, by a 16.9% volume loss in the hippocampus (a temporal lobe structure essential for learning and memory).[140] In a test of general memory, however, these patients were no different from controls.[141] Vestibular signaling to the hippocampus is known to occur in both humans and other primates via a direct, two-neuron

[135] Dieterich M, Brandt T. 2008. Functional brain imaging of peripheral and central vestibular disorders. Brain 131(10): 2538–52.

[136] Hanes and McCollum 2006.

[137] Hanes and McCollum 2006.

[138] Hanes and McCollum 2006.

[139] Brandt T, Schautzer F, Hamilton DA, Bruning R, Markowitsch HJ, Kalla R, Darlington C, Smith P, Strupp M. 2005. Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. Brain 128: 2732–41.

[140] Brandt et al. 2005.

[141] Brandt et al. 2005.

linkage through the posterior thalamus. There are also other proposed neural pathways.[142]

Disordered vestibular input increases error rates in purely mental tasks based on visualization of remembered objects, showing that coherent vestibular input is critical for thinking successfully and efficiently in spatial terms.[143] This is true even without using sight and beyond the period of memory storage. The tasks included detailed visualization, considered an occipital (visual) cortical task, and mental rotation, a parietal cortical task.[144]

Vestibular stimulation in both humans and other primates activates a variety of areas in the parietal cortex, including 1) a core vestibular processing area (posterior insula), 2) the somatosensory strip, 3) areas involved in hemineglect in stroke patients (ventral parietal), and 4) a region "known to be involved in multimodal coordinate transformations and representation of space" (intraparietal sulcus), which is a principal site for arithmetic and counting tasks.[145]

Hemineglect is a condition after right-sided parietal stroke in which a patient can have so much unawareness of the left side of space that he is oblivious to his own left-sided body parts being paralyzed, for example, or undressed. Vestibular stimulation temporarily corrects or improves this unawareness, in ways that suggest stimulation not only to general attention, but also to cerebral structures involved

[142] Brandt et al. 2005.

[143] Mast FW, Merfeld DM, Kosslyn SM. 2006. Visual mental imagery during caloric vestibular stimulation. Neuropsychologia 44(1): 101–9.

[144] Mast et al. 2006. I wonder whether the detailed visualization task also included a parietal component, given the quantitative comparison the subjects had to make with the remembered image.

[145] Hanes and McCollum 2006, p. 82.

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in the mental representation of space.[146,147] Vestibular stimulation also improves hemineglect patients' performance on tasks of visual localization and visual-spatial memory retrieval. At baseline, and again 24 hours after the experiment, their responses were biased away from the left side, but this bias was corrected or improved immediately after left vestibular stimulation.[148]

Studies of hemineglect patients have further shown that many mental operations are "spatialized" and dependent on parietal brain areas that have been lost, including mathematical operations involving a "mental number line" with lower numbers on the left,[149,150] clock representations of time,[151] and spelling at the beginnings (left) or ends (right) of words (errors occur opposite to the side of the parietal lesion).[152] In right-handed patients with right parietal strokes, there is no impairment to simple numeric calculation (a left-sided parietal function), but there is impairment to spatialized mathematical thinking, such as finding the midpoint between two numbers.[153] At the other extreme of mental functioning, it has been found that great mathematicians think of numbers in spatial terms,[154] which "may be more efficient because

[146] Geminiani G, Bottini G. 1992. Mental representation and temporary recovery from unilateral neglect after vestibular stimulation. J Neurol Neurosurg Psychiatry 55(4): 332–33.

[147] Cappa S, Sterzi R, Vallar G, Bisiach E. 1987. Remission of hemineglect and anosognosia during vestibular stimulation. Neuropsychologia 25: 775–82.

[148] Geminiani and Bottini 1992.

[149] Zorzi M, Priftis K, Umilta C. 2002. Brain damage: neglect disrupts the mental number line. Nature 417: 138–39.

[150] Vuilleumier P, Ortigue S, Brugger P. 2004. The number space and neglect. Cortex 40(2): 399–410.

[151] Vuilleumier et al. 2004.

[152] Hillis HE, Caramazza A. 1995. Spatially specific deficits in processing graphemic representations in reading and writing. Brain Lang 48 (3): 263–308.

[153] Zorzi et al. 2002.

[154] Hadamard J. 1996. *The Mathematician's Mind: The Psychology of Invention in the Mathematical Field.* Princeton University Press, NJ. In Zorzi et al. 2002.

it is grounded in the actual neural representation of numbers."[155] A recent study of outstanding human memorizers shows that spatially oriented strategies are also critical to good memory, by providing an efficient framework for memory organization and retrieval.[156]

Thus current research shows that coherent vestibular neural input is critical for spatialized forms of thinking and memory. Spatialized thinking and memory is intrinsic to many of the things we do with our minds, including mathematical thinking and memory organization (as discussed above) and many forms of map-based or visually based problem-solving or short-term memory we do in everyday life. Spatial thinking is used, for example, to figure out the most efficient path for a set of errands, remember the path and images of the items to be obtained, search for the items on the shelf, and judge if one was given the correct change. It is used for mental "maps" or calendars of one's day, week, or month and its appointments, to picture in three dimensions how to put something together, or imagine what has gone wrong inside a device and initiate a repair. It is used, as well, for understanding the visual clues and images in a movie or TV show. In this context, it is easy to see how vestibular disturbance might impair concentration (which means the ability to perform thinking tasks successfully and efficiently) and memory. Vestibular disturbance also has the potential to affect reading directly, via the reflex control exerted by semicircular canal and otolith organs over eye movements (vestibulo-ocular reflex).

Effects on concentration and memory were nearly ubiquitous in the present study, if one includes all subjects that told me about any problems in this area. For some subjects the deficits were

[155] Zorzi et al. 2002.

[156] Maguire EA, Valentine ER, Wilding JM, Kapur N. 2003. Routes to remembering: the brains behind superior memory. Nat Neurosci 6(1): 90–95.

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dramatic compared to pre-exposure baseline, including the 7 out of 10 school-age children and teens who showed a decline in their academic performance. Detrimental effects on concentration and memory were significantly associated with normal memory at baseline ($p = 0.027$) and with fatigue and loss of energy and motivation during exposure ($p = 0.018$). Though sleep deprivation/disturbance undoubtedly plays a role in the problems with concentration and memory, qualitative aspects of the mental performance deficiencies suggest a mechanism other than sleep disturbance alone. I propose that this mechanism is the effect of vestibular disturbance on cognition.

It is interesting here to examine a possible role of vestibular disturbance in the learning of very young children, in the toddler and preschool years. Mrs. G (G2) volunteered that her 2½-year-old's (G5) irritability during turbine exposure was especially triggered by her older siblings' "unsteadying her" or coming so close that she thought she might be unsteadied. Children at this age are learning to keep their balance through a variety of different kinds of activities and postures. They are both fascinated and relaxed by vestibular stimulation (swinging, spinning, rolling, somersaults, etc.) and they actively explore the physical world through this play. The behavior of objects in gravity is another source of fascination, starting with babies' casting behavior and moving on to pouring water, sliding down slides, rolling things down inclines, building dams, floating toy boats, blowing bubbles, releasing helium balloons, etc. Vestibular input and processing play a critical role in a) balance during movement, b) the generation, storage, and use of internal maps, and c) recognition of the behavior of objects under the influence of gravity. Indovina et al. measured brain activity by functional MRI in adults as they watched the movement of simulated objects, finding that the vestibular network was selectively engaged when the acceleration of an object was consistent with natural

dramatic compared to pre-exposure baseline, including the 7 out of 10 school-age children and teens who showed a decline in their academic performance. Detrimental effects on concentration and memory were significantly associated with normal memory at baseline ($p = 0.027$) and with fatigue and loss of energy and motivation during exposure ($p = 0.018$). Though sleep deprivation/disturbance undoubtedly plays a role in the problems with concentration and memory, qualitative aspects of the mental performance deficiencies suggest a mechanism other than sleep disturbance alone. I propose that this mechanism is the effect of vestibular disturbance on cognition.

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gravity, even though the stimulus was only visual.[157] The authors use this as evidence that "predictive mechanisms of physical laws of motion are represented in the human brain"[158] under the influence of vestibular signaling of the vector of gravity. I suggest that these representations of the physical laws of motion are embedded in the human brain during early childhood as toddlers and children learn through experimentation (play) about the behavior of their bodies and other objects in gravity, and that coherent vestibular signaling is critical to this learning.

Environmental noise, learning, sleep, and health effects

Many studies have quantified the effects of environmental noise on children's learning. Reading acquisition—a language-intensive process—is especially sensitive to the effects of noise in school and at home. The effect is distinct from the effects of noise on attention or working memory,[159] and is correlated with measures of language processing such as speech recognition.[160] Airplane noise, which has a large low frequency component, has a stronger effect than traffic noise in some studies,[161] but traffic noise is also shown to have modest effects on memory in quieter communities.[162] Most studies are cross-sectional, but a longitudinal or cohort study, done

[157] Indovina I, Maffei V, Bosco G, Zago M, Macaluso E, Lacquaniti F. 2005. Representation of visual gravitational motion in the human vestibular cortex. Science 308: 416–19.

[158] Indovina et al. 2005.

[159] Haines MM, Stansfeld SA, Job RFS, Berglund B, Head J. 2001. A follow-up study of effects of chronic aircraft noise exposure on child stress responses and cognition. Int J Epidemiol 30: 839–45.

[160] Evans GW, Maxwell L. 1997. Chronic noise exposure and reading deficits: the mediating effects of language acquisition. Environ Behav 29(5): 638–56.

[161] Clark C, Martin R, van Kempen E, Alfred T, Head J, Davies HW, Haines MM, Barrio IL, Matheson M, Stansfeld SA. 2005. Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: the RANCH project. Am J Epidemiol 163: 27–37.

[162] Lercher P, Evans GW, Meis M. 2003. Ambient noise and cognitive processes among primary schoolchildren. Environ Behav 35(6): 725–35.

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when an airport was closed in one location and opened in another, showed similar effects on reading acquisition.[163] One study showed effects of noise on reading and auditory processing in children who lived in an apartment building next to a busy highway. The higher they lived in the building, the quieter were their apartments and the better their reading and auditory discrimination scores (e.g., distinguishing *goat* from *boat*). After controlling for parental education and income, the auditory discrimination scores largely explained the noise-reading linkage.[164] These effects on reading occur at sound levels far less than those needed to produce hearing damage.[165] Children with pre-existing reading deficiencies and children at higher grade levels are more affected, and longer exposure produces larger deficits.[166]

Effects suggestive of wind turbine noise impact on auditory discrimination or central auditory processing were found in the current study. During the period immediately after moving away from turbines and the cessation of her tinnitus, Mrs. A (A2, age 33) found she had a new difficulty understanding conversation in crowded, noisy places. Her son (A3, age 2½) began to confuse several consonant sounds during exposure, and continued to do so in the immediate post-exposure period.

Studies of adults in industrial settings have shown effects of noise on cognitive function when the noise is not considered loud and is nowhere near the threshold for causing damage to hearing. Polish

[163] Hygge S, Evans GW, Bullinger M. 2002. A prospective study of some effects of aircraft noise on cognitive performance in schoolchildren. Psychol Sci 13: 469–74.

[164] Cohen S, Glass DC, Singer JE. 1973. Apartment noise, auditory discrimination, and reading ability in children. J Exp Soc Psychol 9: 407–22.

[165] Evans GW. 2006. Child development and the physical environment. Annu Rev Psychol 57: 423–51.

[166] Evans 2006, p. 426.

researchers exposed workers to 50 dBA broadband noise or 50 dBA broadband noise with low frequency components (10-250 Hz) as they worked on standard psychological tests. Low frequency noise impaired performance more than broadband noise without low frequency components, especially in subjects who rated themselves as highly sensitive to low frequency noise. There was no difference in the annoyance ratings for the two types of noise, nor evidence of either habituation or sensitization.[167]

Sleep deprivation is a primary focus of studies of community noise in general and was a major factor for the subjects in the current study. The occurrence of VVVD contributes a distinctive quality to sleep disturbance and to the extent of sleep deprivation near wind turbines, since waking up in a physiologic state of panic leads to prolonged wakefulness or not returning to sleep at all. A second distinctive quality of wind turbine-associated sleep disturbance was nocturia (getting up repeatedly at night to urinate), mostly in adult women, and nocturnal enuresis (bed-wetting) in a 5-year-old girl. Nocturia resolved immediately when subjects slept away from turbines. For the 5-year-old, the enuresis stopped during a family vacation, resumed on return home, and resolved fully when the family moved away.

Studies of whole-body vibration identify 10–18 Hz as frequencies likely to create the urge to urinate,[168] a possible mechanism for nocturia during exposure. Nocturnal enuresis may be a manifestation of the same direct vibratory stimulation in a child not yet developmentally ready to awaken to bladder signals, or it may instead be a parasomnia (like sleep walking, sleep talking, and night terrors) that occurs during disordered partial arousal

[167] Pawlaczyk-Luszczynska M, Dudarewicz A, Waszkowska M, Szymczak W, Sliwinska-Kowalska M. 2005. The impact of low-frequency noise on human mental performance. Int J Occup Med Environ Health 18(2): 185–98.

[168] Rasmussen 1982.

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from the deeper stages of sleep. Perilymphatic fistula syndrome, a vestibular disorder, includes nocturnal enuresis in adult women in its list of parasomnic manifestations.[169]

Noise at night is known to cause a variety of sleep disturbances, including delay of sleep onset, overt awakening, brief arousals seen on EEG, changes in length and timing of sleep stages, and premature final awakening. Short-term effects of noise during sleep include noise-induced body movements and modifications of autonomic functions such as heart rate, blood pressure, vasoconstriction, and respiratory rate. Noise-induced body movements indicate a low level of arousal from sleep, and occur with noise events as low as 32 dBA. Arousals detected by brain wave pattern on EEG occur with noise events as low as 35 dBA, and conscious awakenings with events of 42 dBA.[170]

Much of the extensive literature on community noise and sleep disturbance focuses on neuroendocrine changes in catecholamine and cortisol levels due to noise disturbance,[171] short-term changes in circulation, including blood pressure, heart rate, cardiac output, and vasoconstriction,[172,173] and the effects of long-

[169] Grimm et al. 1989.

[170] Muzet A, Miedema H. 2005. Short-term effects of transportation noise on sleep with specific attention to mechanisms and possible health impact. Draft paper presented at the Third Meeting on Night Noise Guidelines, WHO European Center for Environment and Health, Lisbon, Portugal, April 26–28. Pp. 5–7 in *Report on the Third Meeting on Night Noise Guidelines,* available at www.euro.who.int/Document/NOH/3rd_NNG_final_rep_rev.pdf.

[171] Ising H, Braun C. 2000. Acute and chronic endocrine effects of noise: review of the research conducted at the Institute for Water, Soil and Air Hygiene. Noise Health 7: 7–24.

[172] Babisch W. 2003. Stress hormones in the research on cardiovascular effects of noise. Noise Health 5(18): 1–11.

[173] Babisch W. 2005. Guest editorial: Noise and health. Environ Health Perspect 113(1): A14–15.

term exposure on the risk of myocardial infarction.[174] There is a significant exposure-response relationship between exposure to nighttime aircraft noise, daily average road traffic noise, and hypertension.[175–177]

Most studies of sleep do not differentiate between low frequency and other types of noise, but there is a growing awareness of the particularly disturbing nature of the low frequency components of community noise.[178] One study compared children sleeping with heavy trucks passing two meters from the house walls every two minutes all night long, to children sleeping with traffic noise without the low frequency component. The low frequency noise–exposed children showed increased cortisol production during the first half of the night (an alteration in the normal circadian rhythm of secretion) compared to the other children.[179] Increased cortisol during the first half of the night was significantly related to restless sleep and difficulties in returning to sleep after awakening during the night.

[174] Babisch W, Beule B, Schust M, Kersten N, Ising H. 2005. Traffic noise and risk of myocardial infarction. Epidemiology 16(1): 33–40.

[175] Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, Dudley M-L, Savigny P, Seiffert I, Swart W, Breugelmans O, Bluhm G, Selander J, Haralabidis A, Dimakopoulou K, Sourtzi P, Velonakis M, Vigna-Taglianti F. 2008. Hypertension and exposure to noise near airports: the HYENA study. Environ Health Perspect 116(3): 329–33.

[176] Eriksson C, Rosenlund M, Pershagen G, Hilding A, Ostenson C-G, Bluhm G. 2007. Aircraft noise and incidence of hypertension. Epidemiology 18(6): 716–21.

[177] Haralabidis AS, Dimakopoulou K, Vigna-Taglianti F, Giampaolo M, Borgini A, Dudley M-L, Pershagen G, Bluhm G, Houthuijs D, Babisch W, Velonakis M, Katsouyanni K, Jarup L. 2008. Acute effects of night-time noise exposure on blood pressure in populations living near airports. European Heart J 29(5): 658–64.

[178] Persson Waye K. 2004. Effects of low frequency noise on sleep. Noise Health 6(23): 87–91.

[179] Ising H, Ising M. 2002. Chronic cortisol increases in the first half of the night caused by road traffic noise. Noise Health 4: 13–21.